CLINICAL LECTURES

ON

DISEASES OF THE HEART

DELIVERED AT

THE MATER MISERICORDIÆ HOSPITAL.

BY

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MITRAL OBSTRUCTION.

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LECTURE I.

MITRAL OBSTRUCTION.

GENTLEMEN,—I propose, in the course of the present session, bringing under your notice in a series of short clinical lectures, the subject of diseases of the heart, confining myself to those forms that have been illustrated by cases in our wards.

This hospital affords an ample field for the study of cardiac pathology, and I trust I may succeed in the endeavour to enlist your interest in the prosecution of it. The first subject I shall take up is that of contraction of the left auriculo-ventricular, or mitral orifice, because it is one upon which a considerable addition has been recently made to our knowledge, and in the diagnosis of which I do not hesitate to affirm, notwithstanding the scepticism of some of our most eminent authorities, that absolute precision has been arrived at, so far, at least, as that is attainable in medicine.

We have had under observation six cases of this form of disease of the heart since December, 1864. In five of these cases the diagnosis of mitral contraction was confidently made; in the sixth, the evidence upon which an opinion as to the state of the heart might be formed was suspended by the extreme debility of the patient, who was not seen by me till a few days before her death with cerebral complication.

In three out of the six cases we have had the advantage of a post-mortem examination, and the morbid specimens obtained are now before you.

I will give a short *resumé* of each of these cases, and

subsequently make a few general remarks upon them, with the view of endeavouring to fix in your minds the most prominent symptoms, and the diagnostic signs, of this particular form of cardiac lesion.

Case 1.—Anne Keogh, an unmarried female, aged 28, a dressmaker, admitted into hospital December 7, 1864. Has suffered for the last few years from palpitation and shortness of breath on making much exertion; these symptoms have latterly become more troublesome. Twelve months previously she began to cough after exposure at night, and for four months preceding the date of her admittance into hospital she has not been able to lie down for more than a minute or two, owing to the dyspnœa which this change of posture occasioned. A month previously her feet began to swell, and about the same time, or somewhat earlier, and repeatedly since, she expectorated some florid blood.

The feet and legs are now much swollen; the face is rather ruddy and not œdematous; pulse 96, weak, small, and irregular; heart acts with great irregularity, every third beat being followed by a rapid "tick tack," the time of recurrence of which, however, occasionally varies; impulse strong and laboured, but not heaving; first sound somewhat rough but well pronounced; second sound double, reduplication being most distinctly audible at right side of apex; chest universally resonant and rather distended, a slight shade of dulness beneath right clavicle; respiratory sound rough, and accompanied with fine crepitant râles all over chest.

Venous pulsation, synchronous with the ventricular systole, of a tremulous character, and arrested by the lightest finger-pressure, is visible in both supra-clavicular fossæ, and also along inner edge of right sterno-mastoid muscle.

December 16th: Breathing much relieved by a large blister; râles less loud; a distinct, somewhat rough, but not loud murmur is heard over left apex *immediately before*, and running up to, first sound ("left auricular systolic

murmur"); no reduplication of second sound to-day; pulse 96, and still irregular at intervals.

17th: Præsystolic murmur audible also, but not so distinctly, over lower part of sternum; not audible over left scapula, where sounds of heart are distinctly heard; pulse 102, occasionally intermitting, and so feeble that patient could scarcely be expected to have strength enough to stand, yet she walks about the ward and corridors, and declares she feels "better;" no lividity of lips or fingers; a good deal of blood-streaked mucus expectorated last night.

31st: Shooting pain in region of heart and extending to left scapula; fremitus over apex; palpitation and breathlessness; pulse 112, small and intermitting; one leech to be applied over heart, and one-eighth of a grain of extract of aconite to be given three times daily.

January 11th: A murmur audible *with* first sound as well as preceding it.

12th: The former, or systolic murmur, *only* audible to-day.

13th: *Both* murmurs again audible. ℞ Extract. acon. gr. $\frac{1}{8}$; pil scillæ c. gr v. M. Ft. pil. One such to be taken three times daily.

14th: Great distension of cervical veins, with respiratory pulsation on right side; cardiac pain shooting to left scapula; great œdema of legs and feet. ℞ Ext. elaterii, gr. $\frac{1}{8}$; pil. scillæ c. gr. iiss.; ext. hyoscyami, gr. i. M. Ft. pil. habeat ii. tales, st. i. 6ta quaque hora.

21st: Neither radial pulse perceptible; considerable œdema of right arm on which she lies; great œdema of legs, which were punctured, and gave off much serum with some relief. ℞ Ext. aconit. alcohol. gr. $\frac{1}{2}$, to be given three times daily in form of pill.

22nd: No pulse to be felt; pain over heart and also in right side posteriorly; base of right lung somewhat dull, with crepitus; systolic murmur *only* audible; second sound reduplicated at midsternum.

23rd: Patient is sinking; no pulse; respirations 18 in

the minute; great venous pulsation in neck; hands icy cold.

24th: Respirations 15 per minute; is unconscious; face livid.

25th: Coughed up some dark clotted blood last night; is still unconscious; respirations 15.

26th: Died at two o'clock this morning.

Autopsy nine hours after death.—Face, trunk, and extremities livid and œdematous. Both lungs emphysematous; lobular apoplexy of both apices, more extensive in right, and also in both bases; in left base a group of emphysematous air-sacs, the size of a large walnut, projects from the surface of the lung, and is filled with dark solidified blood; surface of lungs is slate-coloured in site of apoplectic effusion. About six ounces of serum were found in the pericardium. The heart was greatly dilated owing to distension of its right cavities, which contained a vast quantity of dark currant jelly-like blood; right auricle and ventricle much dilated, and tricuspid orifice so large that the five fingers might be passed through it with ease; the anterior and left segment of the tricuspid valve presented upon its edge several warty growths of minute size but firm consistence; a few vegetations of a similar kind were likewise found on its posterior segment. The walls of the right ventricle were somewhat thickened (being five-sixteenths of an inch); the left auricle was much dilated, and its walls thickened; the pulmonary veins were not dilated. The mitral orifice consisted of a funnel-shaped passage formed by the agglutination of the segments of the mitral valve, and projecting into the left ventricle. This passage is an inch and a quarter long, and so narrow at the end as barely to admit the point of the little finger. The segments of the valve, as likewise the chordæ tendineæ, are much thickened. A fragment of solid fibrin, of a quadrilateral figure, was attached by one of its edges to the auricular aspect of the mitral valve in such a manner that it must have been displaced over

the orifice during the passage of the blood from the auricle into the ventricle.

The left ventricle was contracted and of normal thickness.

The sigmoid valves, both of the aorta and pulmonary artery, are in a healthy state; there is slight atheromatous degeneration of the coats of the aorta above the valves.

In this case the diagnosis of mitral contraction was made and confidently announced on the 16th of December, the day on which the præsystolic apex-murmur was first distinctly recognized.

Dilatation of the right chambers of the heart and tricuspid regurgitation were declared present on the day of the patient's admittance into hospital. Pulmonary apoplexy was identified a short time before the patient's death.

I have given you the details of this case, because it presents a typical example of the particular form of disease of the heart which it exemplifies, with the clinical history of which you should be acquainted. It was submitted to the Pathological Society of Dublin, together with the morbid specimens which are now before you, on the 28th of January, 1865, and will be found fully reported in the *Dublin Quarterly Journal* for November of that year.

Case 2.—Ellen D., aged 30, mother of five children, admitted into hospital January 10, 1865. A fortnight after her last confinement, which took place two months ago, had a "fit," during which she was unconscious, and on recovery found she had lost entirely the use of the right arm, and partially also that of the lower limb of the same side; sensibility is only impaired in both limbs; the mouth is slightly drawn towards the left side; pulse 102, weak, and remarkably irregular, as is likewise the heart's action, which is, however, unattended with murmur.

14th: Second sound of heart doubled.

February 1st: A loud rough murmur immediately precedes the first sound, which is unaffected; second sound double. Patient remembers now that seven or eight years

ago she suffered from palpitation after exertion ; has never had rheumatism, or spat blood ; no œdema in any part of body.

10th : Pulse 84, irregular and failing. Left hospital on 26th February, much improved in general condition, but without any improvement as regards the paralysis. The treatment in this case consisted in the administration of bichloride of mercury and iodide of potassium, with the view of promoting resorption of the extravasated blood, or cerebral embolus, to which the paralysis was attributed ; subsequently strychnia was given in one-sixteenth grain doses three times daily.

Case 3.—Jane L., aged 30, mother of four children, living at Stillorgan, visited the hospital dispensary June 14, 1865, complaining of languor and debility. She is pale ; pulse 96 and " visible ;" respiration natural ; heart's action strong ; loud rough murmur *preceding* first sound, and confined to region of apex ; this murmur occupies latter part of diastolic pause, and ends *at* first sound, which is heard distinctly, unaccompanied by murmur. Second sound is double over apex, where it is loudest, also over base and ascending aorta. One of the junior students present who examined the heart remarked that it seemed to him as if there were " three sounds."

Patient had acute rheumatism, engaging knees and elbows, four years ago, but has no reason to suppose that the heart was then implicated. Seven months ago spat about a pint of florid blood, and a little, which was of a darker hue, three weeks since. Slight râle in left mammary region ; no œdema or dyspnœa.

June 21st : Patient visited the dispensary again to-day ; is much better ; pulse 90, full and regular ; when heart acts strongly, as after exertion, a jarring emitus is felt over apex ; other phenomena as before.

The treatment consisted in the administration of iron and quassia.

Case 4.—Jane Gilligan, aged 34, married, but without children, was admitted into hospital January 26, 1866.

Had rheumatic fever when thirteen years old, but has no recollection of heart having been engaged in that attack, and enjoyed good health subsequently; has occasionally had hæmorrhage from the bowels, which she attributes to "piles." Three years ago received from her husband, whilst under the influence of drink, a blow in the region of the liver, by which she was stunned; suffered pain in this situation, and shortly afterwards became dropsical; has not been jaundiced; the feet and legs were greatly swollen, as was likewise the abdomen; the former were punctured, and after a short course of medical treatment she got quite well of the dropsy, and enjoyed comparatively good health till six weeks ago, when she caught cold from wet and exposure during the voyage from Liverpool to Dublin. Shortly afterwards the feet and legs became swollen, and the breathing oppressed.

On admission, the lower extremities were livid and patchy, and enormously distended with serous infiltration. The toes were purple in colour; some apertures had formed spontaneously in the legs, and from these serum exuded. Pulse small and weak, but regular; orthopnœa; lips livid; conjunctivæ injected, and of a purple tint, with an admixture of jaundice; hands and fingers cold and livid; respiration greatly oppressed; jactitation; urine passed in small quantity not more than three ounces in twenty-four hours, and loaded with bile-pigment.

Owing to the distress which change of posture occasioned, it was impossible to examine the chest posteriorly. It was, however, dull below and in front, and to an extent sufficient to obscure præcordial dulness. Cardiac impulse of ordinary strength, and accompanied with slight fremissement. The apex pulsated in the usual situation, and here two distinct murmurs were heard; the first in the conventional order of cardiac phenomena was loud and rough, and *preceded* the first sound by an appreciable interval; it was limited to the area of the apex, and *succeeded* by the first sound, which was clear and unattended with murmur; the second murmur was less distinct here, softer

and more distant; it was *diastolic* in rhythm, and superseded or replaced the second sound; was loudest at the base, where it was the only murmur distinctly heard, and was traceable upwards in the course of the aorta for about two inches.

Slight disfigurement of hands and fingers from chronic gout, and some serous effusion into peritoneum.

Diagnosis.—Mitral obstruction; partial aortic regurgitation; disease of liver, probably cirrhosis, with effusion into all the serous cavities.

Prognosis in the highest degree unfavourable; death imminent within the next few days.

January 27th: Passed a sleepless night; punctures discharged very little; two patches of gangrene the size of a crown piece, one upon the dorsum of each foot; no radial pulse; great dyspnœa and agonizing pain in the abdomen.

28th: Patient died at half-past one o'clock this morning, having previously thrown up a large quantity of dark coagulated blood.

Autospy ten hours after death by the Resident-Surgeon, C. O'Neill.—Much serous effusion into peritoneum; liver contracted and firm, with thickened capsules, much reduced in volume, somewhat globular in figure, and on section was found to be in the condition described by Kiernan as that of "portal venous congestion." Spleen of average size, firm and heavy; capsules thick and opaque on convex surface. In the substance of the spleen were imbedded several masses of a mortar-like substance, as large as a bean, perfectly encysted, and consisting apparently of lithate of soda. Kidneys healthy; no evidence of peritonitis. Thorax: a large collection of serum in each pleural cavity; lungs healthy, with exception of bases, which were solid and dull on percussion and opaque on surface, owing to thickening of fibrous investment. On section this portion of each lung was firm, dark red, and did no yield either blood or serum. Pericardium contained about

a pint of straw-coloured serum. The surface of the heart was as if universally daubed over with white paint, but polished and glistening; its fibrous envelope was greatly thickened. A flake of white membrane was found floating loosely in the liquid effusion. Heart of average size and consistence. Right auricle contained a large mass of yellow fibrine, ending in a rounded extremity at the auriculo-ventricular opening. Right ventricle of average thickness, somewhat dilated, and containing a few shreds of decolorised fibrine. Pulmonary artery free and healthy. Left auricle somewhat thickened in its walls (see measurements). Left ventricle contracted and thickened. Mitral valve rigid, much thickened, and nearly calcified. The segments were united in such a manner as to convert the auriculo-ventricular opening into a narrow slit-like passage opposing the free entrance of blood into the ventricle, but scarcely admitting of regurgitation. The aortic valves were somewhat thick and rigid, but not in an advanced stage of disease; they permitted the slow entrance of water from the aorta into the ventricle. The lining membrane of the aorta was red and dotted with yellow patches of atheroma.

Measurements of Heart.—Walls of right ventricle 3-16ths of an inch thick at apex, and 3-8ths of an inch in central portion. Right auriculo-ventricular valve consisted of only two segments, the septum of Lieutaud being absent.

Left Auricle.—Cavity $2\frac{1}{4}$ inches from septum to outer wall, $2\frac{3}{4}$ inches from superior wall (near appendix) to root of mitral valve, 3 inches from anterior to posterior wall; thickness of walls in sinus, $\frac{1}{4}$ inch.

Left Ventricle.—Cavity, from root of mitral valve to apex, $2\frac{1}{2}$ inches; antero-posterior diameter near attachments of septum, $1\frac{1}{4}$ inch; thickness of walls, 5-16ths of an inch at apex, $\frac{1}{2}$ inch at central portion, $\frac{5}{8}$ inch at base; aorta 1 inch in diameter immediately above valves.

This case admitted of no hope from treatment, which was, therefore, confined to palliative measures, such as

brandy and etherial stimulants, warm applications to the feet, occasionally small doses of blue pill and extract of taraxacum, with the view of quickening the action of the liver, and liquid nutriment.

The details of the case, together with the morbid specimens, were laid before the Pathological Society of Dublin on the 3rd of February last.

The next case was complicated with right hemiplegia, atrophy of the left anterior lobe of the cerebrum, and loss of speech, and has been fully reported in THE MEDICAL PRESS AND CIRCULAR of May 23, as a good example of aphasia. I will here give only an epitome of the case, dwelling upon those portions of it which have reference to the cardiac lesion.

Case 5.—Jane Quinn, aged 47, married, and the mother of one child, admitted into hospital March 17, 1866. Health has been good, with exception of a few attacks of rheumatism. On the night of the 27th of last December went to bed in her usual health, and on the following morning was found hemiplegic on the right side, and incapable of uttering a word beyond the monosyllables, "yes" and "no;" there was also paralysis of the right side of the face. At the date of admittance her condition had undergone no change as regards voluntary motion in the right side and the faculty of speech; pulse so weak that it was not calculable at the wrist; counted by the heart it was 160; heart's action most irregular, both sounds morbidly clear, extensively transmitted over the chest, and *unattended with murmur.*

Under date of 23rd March, it is reported in my notes that there had been no radial pulse for the two preceding days, and on the following day (March 24th), the action and sounds of the heart had ceased to be perceptible; patient conscious and can swallow liquids; coldness and lividity of extremities, the latter in patches; died at four p.m.

Autopsy twenty-four hours after death.—It is unnecessary

to describe here the condition of the brain, which was of deep interest, and confirmatory of the views of M. Paul Broca as regards the connexion between atrophy of the middle and inferior left frontal convolutions of the cerebrum and loss of speech. The details are given in THE MEDICAL PRESS AND CIRCULAR of May 23rd, and will be published, together with an admirable woodcut of the brain, in the proceedings of the Pathological Society, to which the case was communicated on the 7th April last. The heart was of average size, the left auricle dilated and its walls hypertrophied (see measurements below), and the left auriculo-ventricular orifice much contracted by cohesion of the segments of the mitral valve.

Left auricle one-fourth of an inch thick at superior and left portion; do., one-eighth of an inch thick in central portion; left ventricle, one half inch thick in central portion; do., one-fourth of an inch thick at apex.

The mitral orifice barely admitted the tip of the index finger. The left lung presented a good example of pulmonary apoplexy. The morbid specimens illustrative of the heart and lung complication are now before you, and I will proceed to make a few remarks on this portion of the case.

I entertain no doubt whatever that this poor woman was the subject of endocarditis, implicating the mitral valve, and causing partial cohesion of its segments, in one of her attacks of rheumatism; this was the starting point of her disease, and the immediate cause of her death. On the night of the 27th of December, the woman being then in her usual state of health, a fibrinous embolon was, in all probability, detached from the mitral valve, wafted along by the arterial current, and impacted in the left middle cerebral artery, whence the left anterior lobe of the cerebrum and the upper portion of the left motor tract derive their principal nutrient supply; hence paralysis of motion on the opposite side and loss of articulate speech.

It is true that an embolon has not been found, and

therefore there is not proof that this was the cause of the symptoms mentioned; still, the sudden occurrence of a *local* cerebral lesion, *not* apoplectic, the patient being in her usual state of health up to the time of its occurrence, taken in conjunction with pre-existing valvular disease due to inflammatory deposit on the arterial side of the heart, will admit of no other solution, and affords circumstantial evidence so strong that even in the absence of the peccant body at the period of death, and of a satisfactory explanation of its disappearance, I am forced to assume its existence on the night of the 27th of December, and for some time subsequent to that date; it may have been disintegrated, and have re-entered the circulation in a molecular form; but on this part of the subject I will not further speculate.

The diagnosis of mitral contraction was not made in this case, because the pathognomonic sign of præsystolic apex murmur was not present. This sign, which I believe to be inseparable from mitral constriction, as long as the left auricle contracts with ordinary force, ceases to be developed, as indeed all cardiac murmurs do, some time previous to death, owing to debility of the heart.

In this particular form of cardiac disease it is remarkable, and in my opinion likewise characteristic, how long the patient may exist without a radial pulse. Thus, in Case 1, I find the following remark in my notes on this subject, under date December 17, *forty days* before the patient's death: "Pulse 102, and so weak the wonder is she is able to stand; yet she walks about the wards and corridors quite firmly, and declares she is better." On the 21st of January neither radial pulse was perceptible, nor was it to be felt at any time subsequent to this date, although the patient lived till the 26th—that is, for a period of *one hundred and twenty hours*, with absolutely no pulse at the wrist.

In the case now under consideration (Case 5) the radial pulse was so weak on the 17th of March (the day of admittance) that it could not be registered; on the 21st, and after that date, there was no radial pulse, yet the

patient lived till four p.m. on the 24th—*i.e.*, *seventy-two hours* after pulsation at the wrist had ceased.

The preceding considerations may afford an explanation of the absence of præsystolic murmur in a case of veritable mitral contraction, seen only during the more or less protracted period of profound debility immediately preceding death.

The other signs and symptoms characteristic of mitra contraction were present in the case under notice, but obscured by the more prominent symptoms arising from the cerebral lesion; thus, the pulse was flickering and intermittent, and ultimately failed altogether some time before death; there was comparatively little venous engorgement or lividity of the surface or extremities, such as are witnessed in patients suffering from mitral regurgitation, for the obvious reason that the circulation by the arteries being in defect, the systemic veins were consequently not surcharged with blood, as they are in the last-mentioned form of disease. This difference is probably due to the fact, that in mitral regurgitation the left ventricle is in a state of hypertrophy, and therefore acts with compensatory force upon the column of arterial blood escaping by the aorta, whilst, at the same time, it exercises an unwonted back pressure upon the pulmonary circulation through the patent mitral orifice, and thus upon the venous side of the heart; whereas in mitral contraction left ventricular hypertrophy does not exist, and mitral patency, if at all, only in a moderate degree. Consequently the pressure upon both ends of the circulating column is much less, and the volume of blood escaping by the aorta being already much reduced, the pulse is small and faltering, and reflux upon the lungs, and through them upon the right side of the heart, is less in quantity and in force.

For the reasons just given respiration is much less embarrassed, and dropsical effusion less general and less considerable in mitral obstruction than in mitral regurgitation, but whatever the cause may be, the fact is as now stated.

The action of the heart in cases of mitral contraction is likewise peculiar; it is quick, weak, markedly irregular, and of the "tick-tack" character; and the sounds are sharp and propagated to a great distance over the chest. The second sound is also not unfrequently *reduplicated*; this occurred in at least two of the cases in the present category. It is not easy to explain this phenomenon; the most rational view consistently with our present knowledge is that which attributes it to a want of synchronism in the closure of the aortic and pulmonic valves. In mitral contraction the great difference in tension of the aorta and pulmonary artery may cause a corresponding difference in the period of closure of the two sets of valves.

The last case I shall submit to you has been recently under your observation, and therefore you will remember all the particulars connected with it.

LECTURE II.

MITRAL OBSTRUCTION.

Case 6.—Mary S., aged 26, a native of the county of Clare, admitted into hospital, May 4, 1866. Seven years ago she emigrated to the United States of America, and whilst there had several attacks of rheumatism, in one of which the heart was engaged, as may be inferred from blisters having been at that time applied to the præcordium.

She returned to Ireland in July, 1865. About a year ago her legs became swollen; the swelling disappeared after a short time, but it has since occasionally returned. A month previous to the date of admittance she threw up a quantity of blood, and again a fortnight later.

Present condition.—Pulse 72, weak, but regular; tongue clean; appetite bad; bowels free; face fresh-looking and free from congestion; œdema of feet; chest universally resonant, and respiration natural; dyspnœa on exertion; area of præcordial dulness not increased; apex beat feeble, and in normal situation; here both sounds are distinct, the first a shade softer and more prolonged than natural, but unattended with murmur. *Immediately preceding*, and extending to the first sound, is a soft bellows murmur, occupying the latter portion of the diastolic

pause, and perfectly distinct from either normal sound. This murmur is loudest at the apex; it is faintly audible at the base; not at all in the course of the aorta, but distinctly audible at the inferior angle of the left scapula. The second sound is greatly intensified over upper edge of left third costal cartilage, in the area of the pulmonary artery. To have chlor. ether and aromat. spirit of ammonia, of each ʒiii., in camphor mixture to eight ounces; a tablespoonful to be taken three times daily; nutritious diet, fresh air, and very gentle exercise.

May 15th: A murmur *accompanies* first sound to-day; no murmur precedes it.

May 16th: Condition is much improved; no œdema of feet; pulse now strong, as well as regular; appetite and sleep satisfactory; no systolic murmur audible to-day; præsystolic murmur distinct as on the day of admission. Patient discharged at her own request, as she declares she feels well enough to resume her duties as house-servant.

This was by no means an aggravated case of mitral contraction; or rather the disease was, in this instance, in its earliest stage, after the subsidence of the acute inflammation which gave rise to it. The symptoms and signs were, however, quite unmistakeable; thus, the rather fresh tint of the face, associated with a quick and thready (though in this instance, not irregular) pulse; œdema of the feet; slight dyspnœa on exertion; expectoration of blood, with scarcely any cough, and none of the signs or symptoms of phthisis; and above all, the characteristic murmur preceding the first sound of the heart, and loudest at the apex, left no doubt whatever upon my mind as to the precise nature of the cardiac lesion to which they were due.

Since I commenced to prepare this lecture another case (being the seventh) of mitral contraction has come under our notice; it is now in St. Catharine's ward, and you have an opportunity of studying and applying to it the diagnostic rules which I have laid down for your guidance.

Case 7.—Jane Maguire, a poor woman, remarkably thin,

aged 50, was admitted June 1, 1866; health has been moderately good; has never had rheumatism; two years ago, after suffering much mental distress, she experienced, for the first time, palpitation and pain in the region of the heart; latterly her feet and legs became swollen; pulse so weak and irregular that it cannot be counted at the wrist; it is quite imperceptible on the right side, and barely perceptible on the left; counted by the heart it registers 150 in the minute; liver is much enlarged in both lobes, and descends nearly to the umbilicus; it is prominent but even on the surface, and not tender on pressure; there is not, nor has there been, jaundice; slight œdema at outer ankles; fingers and toes cold and livid; face rather fresh in colour and not indicative of cardiac distress; heart's action remarkably irregular; both sounds clear and ringing, unattended with murmur, and extensively transmitted over front of chest; especially loud in course of aorta; cardiac impulse strong and labouring, contrasting markedly with radial pulse; apex-beat diffused, but not towards left side; resonance percussion and respiratory sound over lungs are normal; some mucous râles over base of right lung. To have a rhubarb draught and two ounces of whisky with nourishment.

June 2nd: Pulse barely perceptible on right side; somewhat stronger on left, and 84 in the minute. Patient feels better. To have a tablespoonful of the following three times daily:—℞ Tinct. ferri sesquichlorid. ether. chloric. aa. ʒii., infusi quassia ʒviiss. M.

June 10th: Much œdema of feet and legs; orthopnœa; dulness over base of left lung, and here coarse crepitant râles are heard. To be dry-cupped over lower part of left side of chest, and take the following, viz.:—Tinctura digitalis gtts. v., spirit. juniper. c. ʒi. in mist. camphoræ ʒi. every sixth hour.

June 12th: Passed a bad night, having slept scarcely at all. To have chloric ether ʒi., solution of muriate of morphia ℥ii., in an ounce of camphor mixture every night.

15th: Pulse not perceptible on right, and barely percep-

tible on left side; it is irregular and intermitting, as is likewise heart's action. A *systolic* "whiffing" murmur is audible over the apex, or rather somewhat to the left of the nipple to-day; it is not audible elsewhere; both cardiac sounds are heard at the inferior angle of the left scapula, but no murmur. Second sound is much intensified over root of pulmonary artery; dulness on percussion over base of left lung, where respiration is feeble and attended with crepitus; spat some blood mixed with mucus last night; œdema of lower limbs; face free from œdema and lividity; no venous engorgement of neck; very little respiratory distress, save at night; a stinging pain occasionally experienced in region of heart, which has been relieved by an opiate plaster.

June 19th: Pulse 108 counted by the heart; orthopnœa; scarcely any respiratory distress in sitting posture; both sides of chest dull inferiorly, and to a corresponding extent respiration is weak and accompanied with obscure crepitus; has continued to expectorate a little blood since last report; an obscure rubbing or grating murmur immediately *preceding* the first sound is heard over the apex; it occupies the latter portion of the long or diastolic pause, but does not touch the first sound. For the last few days, having a strong impression on my mind that this case was one of mitral constriction, owing to the peculiar grouping of signs and symptoms so closely resembling those of the cases already detailed, I had been looking out for the characteristic præsystolic apex murmur, and mentioned my suspicions to the class. To-day, for the first time, this murmur was distinctly heard and identified, and thus the evidence of mitral obstruction was rendered complete.

June 21st: Pulse 114; respirations 30; urine 1015 s.g., acid, and free from albumen; murmur is to-day systolic exclusively.

June 22nd: Præsystolic murmur alone audible to-day; it is rough but faint, and audible only occasionally—*i.e.*, when, as happens once every two or three minutes, the heart acts several times with unusual force and rapidity.

June 23rd: Præsystolic murmur remarkably distinct to-day, and is the only murmur present; it is not audible in back, nor is second sound accentuated over pulmonary artery.

Before attempting to estimate the diagnostic value of the *præsystolic* apex murmur, as evidence of obstruction at the left auriculo-ventricular orifice, it is necessary you should have a definite and correct idea of the natural order, grouping, and rhythm of the phenomena which constitute a cycle of the heart's action.

As I cannot make this portion of our subject clearer than by quoting from my paper on "The Rhythm of the Heart's Action," read before the Medical Society of the College of Physicians on the 19th May, 1865, I will take the liberty of doing so briefly:—

"The whole period covered by a single action of the heart is divisible into two parts of unequal length, corresponding to the *ventricular systole* and the *ventricular diastole*. Of this period one-third is occupied by the ventricular systole, and two-thirds by the ventricular diastole, the pulse being at the rate of 90 in the minute.

"The first sound and the impulse initiate the ventricular systole, and are immediately succeeded by a short period of silence, or a pause, during which the ventricular systole is continued; this pause, which corresponds to the 'short pause' of French writers, I propose to distiuguish by the title of 'systolic pause,' as indicating the state of the ventricles during its occurrence; it is of very brief duration, at the normal rate of the heart's action, not calculable, but distinctly appreciable by the ear, and is a pause only in the sense that that portion of the ventricular systole which coincides with it is unattended with sound.

"The second sound immediately succeeds the systolic pause, and determines its duration; the period of time covered by it is quite incalculable, but belongs to that assigned to the ventricular diastole. It is succeeded by a

long pause, which I propose to designate as the 'diastolic pause,' in contradistinction to the former.

"This pause corresponds to the interval between the second sound and the first, and, at a pulse-rate of 90, occupies a period of one half second, *minus* the length of the second sound. During this pause the ventricles are undergoing dilation and the auricles contraction. There is, therefore, no pause in the sense of complete absence of movement of any of the chambers of the heart; the so-called pause is a period of *silence*, not one of inaction.

"During the period corresponding to the second sound and the diastolic pause—that is, to the ventricular diastole—the auricles are in a state of contraction; this is feeble and of an undulatory character till towards its conclusion, when it becomes quick and energetic, completing the distention of the ventricles, and is immediately succeeded by their contraction. This terminal portion, or acme, of the contraction of the auricles I propose to designate the 'momentum of the auricular systole,' because then the auricles, having discharged a portion of their contents, have acquired their maximum power of contraction, and propel a further volume of blood with increased force and rapidity into the ventricles.

"The forced and sudden entrance of blood into the ventricles during the momentum of the auricular systole serves the useful purpose of communicating to the ventricles the stimulus of distention, in response to which they immediately react upon their contents, and thus complete the cycle of the heart's action."

A murmur occurring during the period of the first sound is usually designated a "systolic murmur," and a murmur occurring during the period of the second sound a "diastolic murmur"—that is, *ventricular* systolic and diastolic respectively. This nomenclature would be unobjectionable did the ventricular systole accurately corresponding in duration with the first sound, and the ventricle diastole with the second sound. Such, however, is not the

case ; for the ventricular systole, as already shown, extends over the first sound *plus* the systolic pause, and the ventricular diastole over the second sound *plus* the diastolic pause. Hence, it should be distinctly understood that the terms "systolic" and "diastolic," as now commonly applied to cardiac murmurs, are intended to convey that the murmurs spoken of *coincide with*, or *replace*, the normal first and second sounds respectively. With this limitation the terms are quite unobjectionable, and may be allowed to stand; but without it they must lead to confusion and laxity in diagnosis.

The application of the terms "systolic" and "diastolic' being thus restricted to what they are really meant to express—namely, murmurs coinciding with the sounds of the heart—it is manifest that other designations must be found for murmurs which are *out of time* with these sounds. To this class of murmurs belongs, in a preëminent degree, that which has been named "præsystolic," and by Gairdner "left auricular systolic;" the former term implying that the murmur *precedes* the first sound which inaugurates the ventricular systole, and the latter, that it coincides in time with the contraction of the left auricle.

If the limited application of the term "systolic," already suggested, be borne in mind, the designation "præsystolic," as applied to this murmur, will be strictly appropriate, sufficiently expressive of its rhythm, and the most convenient for use.

I prefer this to the title "auricular-systolic," which only expresses that the murmur coincides with the contraction of the auricle without indicating the portion of that contraction which it corresponds.

It has been already shown that the contraction of the auricles coincides with the second sound and the succeeding pause. A murmur may be, therefore, in strict propriety of language, "auricular-systolic," which coincides with the second sound, or immediately succeeds it; yet such a murmur would not be that intended to be expressed

as indicative of mitral obstruction, but rather one of very common occurrence and having its seat at the aortic orifice.

The pitch and quality of this, as of other cardiac murmurs, will be found to vary in different cases. It is, however, usually harsh and whispering, thus, rrrr fine; in time it occupies the terminal portion of the long or diastolic pause; immediately *precedes* the first sound, running up to, without extending into it, and therefore coincides with the acme or "momentum" of the auricular systole.

A murmur of this character and rhythm, loudest at the left apex, not audible, or faintly so, at the base, and not transmitted save occasionally to the left side of the lower dorsal spine, may be regarded as diagnostic of mitral obstruction; and if, to the physical evidence thus afforded, be added œdema, rarely and never till the last stage of the disease extending to the face; comparatively trivial venous engorgement; shallow respiration, dyspnœa, and occasional hæmoptysis, not due to primary disease of the lungs; and lastly, the thread-like, faltering, irregular, and intermitting pulse, the evidence is now complete, and fully warrants the confident diagnosis of narrowing or stenosis of the mitral orifice.

Taken alone, and with the limitations as to site and rhythm already mentioned, the murmur now described may be considered pathognomonic, but without these qualifications a localized attrition murmur due to pericarditis, and possibly also a murmur caused by patency of the foramen ovale, or by an aneurism of the left auricle, may be mistaken for it.

None of these murmurs, however, would have its point of maximum intensity at the left apex, or be inaudible at the base. Nor, in the absence of mitral constriction, would any of them be associated with the remarkable group of symptoms already described as characteristic of this lesion.

It is right I should inform you that stethoscopists are by no means agreed as to the identification and significancy of the præsystolic murmur; some deny it altogether, and

many, whilst admitting, attribute it to causes other than mitral obstruction.

It is no part of my duty to reconcile or explain these discrepancies; I am content to reassert in the most confident manner all I have advanced in relation to this murmur, and to express my conviction, notwithstanding the scepticism or dissent of men for whose opinions I have the greatest respect, that ere many years it will have assigned to it a prominent place in the catalogue of acoustic signs which yield definite and positive evidence of special structural lesions of the heart.

Dr. Blakiston ("Diseases of the Heart," 1865, page 246) says, "The chief physical sign of mitral obstruction is a diastolic murmur; it is rarely, however, that such a murmur is engendered, because the size of the auriculo-ventricular opening is so large," &c. Here the murmur of mitral obstruction is described as "diastolic;" this is not strictly correct in the conventional sense, and by fixing attention exclusively on the second sound may lead either to non-observance of the murmur where it exists, or to non-identification of its true nature even when detected.

Præsystolic murmur is, no doubt, frequently absent in cases of veritable mitral obstruction, but never till the advanced stages of the disease, when the vigour of the heart has become much impaired, and even then *only at intervals*, except as regards the period immediately preceding dissolution.

Dr. Peacock ("Valvular Diseases of the Heart," 1865, page 109) says, "In cases of mitral disease it would often be difficult, if not impossible, to distinguish between simple obstruction and regurgitation;" and Dr. Andrews, in his recent article "On the Diagnosis of Systolic Endocardial Murmurs whose point of greatest Intensity is at or near the left Apex of the Heart," published in the first volume of "The St. Bartholomew's Hospital Reports," October, 1865, writes, "At the same time it must be confessed that the diagnosis of a præsystolic from a systolic ventricular

murmur is one of the most difficult tasks in the physical examination of the heart, and is often all but impossible.

I have thus collated the opinions of three of the most recent writers on diseases of the heart, in order that you may understand the present state of knowledge on this subject, and have your senses sharpened for further observation.

Skoda says (Markham's translation, page 232), "In the left ventricle during its diastole a sound unaccompanied by a murmur—second sound—indicates that there is no constriction of the left auriculo-ventricular opening, and that the blood, in passing from the left auricle into the left ventricle, does not flow over any roughened surface."

In reference to this passage I will only observe that the diastole of the ventricles commences *with* the second sound and ends *at* the first; that the only sound occurring within this period is the second, which is never *accompanied* by murmur in simple obstruction at the mitral orifice, whether constrictive or not; and that, therefore, the absence of murmur with the second sound affords no evidence whatever of the absence of disease at the left auriculo-ventricular opening. The murmur indicative of mitral constriction or roughening occurs in the latter portion of the long or diastolic pause, in the "præsystole" of Gendrin, and is therefore preceded and separated from the second sound by the first and greater portion of that pause.

In the *Edinburgh Monthly Journal* for January, 1854, Dr. W. O. Markham gives a very good account of this murmur under the name of "Diastolic Mitral Murmur," with illustrative cases, in which the principal features of obstruction at the mitral orifice are clearly set forth; he describes the murmur as immediately preceding the impulse of the heart, and as being indicative of mitral obstruction.

In the first edition of his work on "Diseases of the Heart," published in 1856, Dr. Markham also devotes a few pages to the discussion of this subject, and strongly urges the diagnostic value of the "diastolic mitral mur-

mur;" to him, therefore, is undoubtedly due the merit of having been the first writer in these countries to draw attention to the subject.

To Dr. W. T. Gairdner of Glasgow, however, the profession is chiefly indebted for a full and satisfactory exposition of the differential diagnosis of mitral obstruction.

In his original and admirable work, "Clinical Medicine," 1862, page 599, Dr. Gairdner says, "Auricular systolic murmurs are certainly not rare; for here are seven of them, at least, in three months, as a counterpart to the same number of aortic murmurs, which nobody will assert to be rare. To me they are among the commonest and the most easily detected of all the cardiac murmurs; and, seeing that I regard the auricular-systolic murmur as all-important in the diagnosis of primary mitral disease, you may trust me when I tell you that in many years' hospital experience I have not seen a single instance in which an auricular-systolic murmur, being of mitral origin, has been produced by *mere* regurgitation, not a single instance in which such a murmur has occurred without either vegetations or contraction of the orifice. On the other hand, I have seen many cases of widely-dilated mitral orifice with evident regurgitation, but without obstruction or deformity; and in every one of these cases the murmur (if present at all) has been ventricular systolic. In our seven cases, therefore, I feel as sure as I can well be of anything in medicine that we have to do not only with mitral disease, but with mitral obstruction." I willingly and cordially subscribe every word of this passage, with one reservation—I do not think the præsystolic murmur easy of detection; it requires close attention, and a readiness in seizing and appreciating the peculiar and distinctive *rhythm* of the murmur, and therefore considerable practice, to detect it in the majority of cases.

Dr. Gairdner's cases were seven in number, as published in the "Clinical Medicine," amongst which were two deaths and one dissection, the latter confirmatory of the diagnosis.

My cases are also seven in number, with three deaths and three dissections; in six out of the seven the diagnosis was made; in the seventh it was not made for reasons already given, although post-mortem evidence showed that mitral constriction was considerable. In the two remaining cases in which a post-mortem examination was made it was confirmatory of the diagnosis.

The prognosis in these cases is of the most unfavourable character. I cannot altogether agree with Dr. Gairdner in the opinion that "mitral regurgitation on the average of cases is a much more immediately dangerous form of disease than mitral obstruction;" it may be more "immediately," but is not more certainly fatal than mitral obstruction.

In regard to treatment very little need be said. The tendency to death is by asthenia and vascular engorgement of the lungs; the former due to failure of the systemic circulation by defect of supply, and the latter to obstruction on the left side of the heart. The object of treatment should therefore be to sustain the failing circulation by increasing the contractile force of the heart, which probably has itself suffered some impairment by defective coronary circulation.

This is best accomplished by the administration of ethereal and alcoholic stimulants, and by nutritious diet frequently given, and in a concentrated form.

Congestion of the lungs should at the same time be kept in check by means of dry cupping and counter-irritation, and the *volume* of the blood kept at a low standard by mild hydragogue aperients and diuretics. I have found much benefit in the treatment of these cases from the use of tincture of the sesquichloride of iron and chloric ether, of each three drachms, in seven ounces of infusion of quassia; a tablespoonful to be taken three times daily.

I have now done, and for the length to which my observations have been extended, the interesting nature of the subject, its novelty amongst us, and my desire to put you in possession of the clinical history and distinctive

characteristics of these cases, and of the present state of knowledge on the subject generally, must be my apology. I shall continue to use such opportunities as may be presented to me of making further observations on this subject, and at some future time I hope to lay the result before you.

July 2nd: Since the last lecture was delivered, the patient, Jane Maguire, whose case is given as the last in the category, has died. For several days she had been somnolent, occasionally delirious, and expectorating large quantities of dark grumous blood; the radial pulse had entirely failed; and on Friday, 29th June, she sank quietly at two p.m. The post-mortem examination, made in presence of the class, and of which I subjoin a summary, was entirely confirmatory of the diagnosis. I may remark here that whilst in hospital this patient was examined by several medical gentlemen, and was repeatedly pointed out by me to visitors and to the class as affording a typical illustration of stenosis of the mitral orifice.

The liver was contracted transversely, and elongated vertically, and in a condition evidently the result of tight lacing in early life, but otherwise healthy; kidneys and other abdominal organs healthy; both pleuræ and also pericardium nearly full of serum; right lung studded with masses of extravasated and solidified blood, varying in size from a pea to a pullet's egg; left lung presented only a single such collection, of medium size, near its anterior edge; heart enlarged, weighing $12\frac{3}{4}$ oz., with one inch of the aorta and pulmonary artery attached; right ventricle dilated and thickened, and containing a thick flake of yellow fibrine, which extended into the pulmonary artery; left auricle greatly dilated, and its walls much hypertrophied; pulmonary veins likewise much dilated; left ventricle hypertrophied, but scarcely dilated; right auriculo-

ventricular orifice much dilated, admitting four fingers with ease; left auriculo-ventricular orifice greatly contracted, and barely admitting the tip of the index finger passed from the auricle; both segments of the mitral valve were greatly thickened, of cartilaginous consistence, but flexible, white, and perfectly smooth on the surface and edges; when water was poured into the ventricle, the aorta being closed by compression with the fingers, the segments of the mitral valve fell together evenly, and prevented regurgitation into the auricle; the anterior and right segment of the valve was the thicker of the two, and one of the chordæ tendineæ extending from it to the anterior wall of the ventricle had become as thick as the point of the little finger; the aortic valves were perfectly healthy, and retained water.

Thus, then, this case being included, the post-mortem examinations were four in number, and in three instances confirmed the diagnosis. In the fourth instance (the case of aphasia) the diagnosis of mitral obstruction was not made, for reasons previously given; but this only shows that the evidence relied upon as warranting the differential diagnosis of obstruction at the mitral orifice occasionally fails, where serious complication exists to mask or derange it, and still more frequently in the period of cardiac asthenia, of more or less protracted duration, immediately preceding death; it does not in any measure diminish the positive value of that evidence when present, and the circumstances under which a diagnosis cannot be arrived at from want of the usual and distinctive signs and symptoms —namely, those of impending death—render it of very little consequence whether the disease be identified or not.